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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Observations Pertaining to Electric Shock Therapy

(Somatic Manifestations)

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Colonel M.C., U.S.A.

This report is based on a study of 162 patients in various diagnostic categories who received a total of 1,590 electric shock treatments. The term "somatic" is used here, as it was by Himwich,[1] to exclude autonomic or visceral manifestations. All treatments were given with standard stimulus apparatus. The duration of current flow was always five seconds with a peak milliamperage of from 550 to 650 at 0.3 milliseconds. The electrodes were held in place manually on either side of the head in such a way that an imaginary line between the centers of the two electrodes would pass through the frontal lobes just anterior to the motor area. No record was made of which was the active and the ground electrode. It is assumed that the path of the electric current through the head was similar to that described by Hayes[2] and Smitt and Wegener.[3]

The reactions observed are not statistically significant because as the study progressed new observations were made from time to time and until a particular phenomenon was noted it was not watched for. For example, the number of patients who had spontaneous positive Babinski reactions cannot be reported because this reaction was not noted until it happened to be observed after the study was well under way.

Somatic Manifestations During the Convulsion

When the treatment went according to plan, the patient was instantly rendered unconscious as soon as the electric current began to pass through the brain and simultaneously with the onset of unconsciousness, the patient went into a generalized tonic convulsion. When the current was broken at the end of five seconds, there was frequently an accentuation of the intensity of the convulsion. A few seconds after the current ceased to flow, the convulsion changed from a tonic phase to a clonic phase.

During the tonic phase of the convulsion, the eyelids were closed tightly and this tonic spasm of the eyelids changed to a blinking of the eyelids as the patient went into the clonic phase of the convulsion. There was almost always conjugate deviation of the eyes during the clonic phase and the direction of the deviation varied from one patient to the next but since no record of which was the active electrode was made, we have no explanation of this variability. Subsequent observations on ten patients indicate that the direction of deviation is not related to the right or left placement of the acting electrode. The jaw muscles went into tonic spasm and this tonic spasticity ordinarily continued even after the clonic phase of the convulsion had subsided. Some patients extended their heads on their shoulders with their neck muscles in tonic spasm while others flexed their heads. In both instances, tonic spasm of the neck muscles continued beyond the cessation of the clonic phase.

Patients were routinely treated with their arms adducted against the chest wall and with their forearms folded in 90° flexion across the abdomen. During the convulsion,

most patients adducted their arms, maintained their forearms in rigid 90° flexion and flexed their wrists and fingers. The violent flexion of the fingers sometimes resulted in the patients' scratching themselves or an attendant who was holding the hands. Occasionally a patient who had a very severe convulsion would extend the arms.

The trunk sometimes assumed an opisthotonoid position during the convulsion but usually the spine was flexed and the shoulder girdle was raised off the bed. All patients were treated with spine in hyperextension.

Most patients maintained their thighs in adduction and extension, their legs in extension and their feet and toes in plantar flexion but some flexed their thighs and legs during both the tonic and the clonic phase of the convulsion. Babinski reactions frequently occurred spontaneously bilaterally.

The motor phenomena described above are characteristic of those listed by Fulton[4] as being the signs of decorticate or high decerebrate rigidity in man.

Stages of the Postconvulsive Awakening Reaction

The observations reported agree with those of Lowenbach and Stainbrook[5] that the electrically induced convulsion leaves a human being in a state in which all that is called the personality has been extinguished. While the patients are in this postconvulsive state, which we shall hereafter refer to as *the first stage of the awakening reaction,* the heart is beating and the patient is breathing. Sometimes tendon reflexes were exaggerated, sometimes they were diminished and sometimes they could not be elicited. Ankle clonus was sometimes present but at other times it was absent. More often than not, the plantar reflex was flexor although a number of patients temporarily showed extension of the great toe and definite fanning of the other toes. The corneal reflex was usually diminished or abolished for a short period following the convulsion. The pupillary light reflex was difficult to evaluate due to certain autonomic manifestations.

Many patients showed apneusis when they first began to breath, i.e., there was marked inspiratory spasm and inspiration was very deep and prolonged. Apneusis occurred only in those patients who had been rendered deeply unconscious and were slow to awaken. In addition to the apneustic breathing, these patients had prolonged spasm of the muscles in the back of the neck and their forearms were flexed in plastic spasticity. This is of interest in connection with the report by Breckenridge, Hoff and Smith[6] that apneustic breathing is a respiratory counterpart of decerebration done at the mid-pontine level. These authors concluded that apneustic breathing in decerebrate animals is a result of the removal of inhibitory influences which normally keep the reticular facilitatory system in check.

Following this relatively inactive period, some patients passed from unconsciousness into sleep and engaged in no somatic motor activity. Most patients, however, went from the first stage of relative inactivity into what Hemphill[7] designated as the stage of involuntary movements, or *the second stage of the awakening reaction.*

One of the most commonly observed involuntary activities consisted of sucking movements and sounds similar to those made by an infant nursing an empty breast or bottle. Sucking was usually the first activity the patient engaged in after beginning to breathe. This is of interest in connection with the report by Babkin and Van Buren[8] of a "center of deglutition" in the medulla oblongata. Occasionally this sucking activity was accompanied or followed by biting. When the nurse removed the mouth gag from one patient following three separate treatments, the patient instantly grabbed the gag away from the nurse, put it back into her mouth and chewed it violently. Another patient would take the mouth gag in her own hand and tear it into small bits with her teeth. Such activities as these suggested the possibility that what are being called involuntary movements may, in fact, be voluntary but on an unconscious level. The terms *consciousness* and *unconsciousness* are used with a realization that they are relative terms and that it is most difficult to adequately define them.[9]

Many patients engaged in what appeared to be purposeless movements during the second stage of the awakening reaction. These included turning the head from side to side,

movements of the hands back and forth across the chest, alternating flexion and extension of the lower extremities and rolling of the trunk from side to side. The purposeless activity did not appear to be accompanied by any emotional experience but had the appearance of a generalized purposeless discharge of energy through muscular activity. Several patients picked at the bedclothes and at their own clothing in a manner reminiscent of carphologia. Other patients carried out hemiballistic movements with their upper extremities.

A considerable number of patients, particularly those being treated for pain for which no organic cause could be found, engaged in activities resembling those described by Cannon[10] as manifestations of sham rage. These patients appeared to be angry, they became vulgar and profane and attacked those present in the room so that they had to be forcibly restrained. This period of sham rage almost invariably passed suddenly into a stage in which the patients were unduly obsequious. As an example, one patient suddenly ceased threatening the author and said a child's prayer, ended by a request for the Lord to bless the author.

A number of patients, most of whom gave a history of varying types of fears as symptoms of their illnesses, showed by the expression on their faces and by their actions during the second stage of the awakening reaction that they were experiencing intense fear. Some of these patients screamed and attempted to escape from the room. Others screamed but instead of actively attempting to escape, laid in the bed and performed movements of their lower extremities resembling running and pedaling a bicycle.

Many patients engaged in sexual activity during the second stage of the awakening reaction. Female patients performed writhing movements of their bodies and lower extremities, flexed their thighs and legs, manipulated their genitalia, became flushed in the face, breathed rapidly, made grunting sounds and sometimes actually appeared to have an orgasm followed by a period of contented relaxation. Male patients were never observed to assume the prone position and execute the movements characteristic of sexual intercourse. Instead they manipulated their genitalia, became flushed in face, breathed rapidly and sometimes masturbated until they ejaculated. Most of the patients who gave a history of overt or latent homosexuality engaged in sexual activities characteristic of the opposite sex. One male in particular used both hands to make a fold in his trousers resembling the female external genitalia and then actively executed female masturbatory movements. Several female patients, and one male patient who indulged in female sexual awakening reaction activities habitually turned suddenly from the supine position to the prone position and as soon as becoming prone, looked up and smiled.

Some schizophrenic and hysterical patients engaged in bizarre and sometimes cataleptic activities. None of the hysterical patients who had these bizarre awakening reactions were completely cured by the shock treatments and, in general, the more bizarre the awakening reaction of the schizophrenics, the less favorable was the response to electro-convulsive therapy.

One patient habitually executed the movements of a child playing pat-a-cake during the second stage and if her physician held out his hands, she played pat-a-cake with him. This same patient, following her last treatment (resulting in a cure of over one year's duration) curled up in the fetal position. Other patients assumed the fetal position near the end of the course of treatments.

None of the patients could recall any of these activities upon regaining consciousness and even when told what they had done, still were unable to remember.

The third stage of the awakening reaction is the stage of self-awareness. At the time when the patient responded to the question, "How do you feel?" it was considered that the patient had entered the third stage. Interestingly enough, almost without exception, the patients responded to this question by stating that they felt fine regardless of how terrible they felt prior to the treatment. At this point, it seems appropriate to point out a type of fear reaction which occurred just as patients went into the third stage of the awakening reaction. These patients, in response to conversation or activity in the room, would suddenly have a startle reaction

which was usually followed by a smile and the statement, "You frightened me." Some patients at this point knew their names and recognized their physician but most patients did not recognize their physician and stated that they had never seen him before. Married women at this point frequently gave their maiden name. Some patients in response to repeated questioning as to their age would, at first, give it as less than ten, then in the teens and subsequently in the twenties and thirties. Some patients showed evidence of motor aphasia before they were able to answer questions. During the third stage (especially if the patient had had as many as three or four treatments) there was frequently a loss or diminution of the inhibitions acquired in our culture. Patients were careless about exposing their bodies and some patients used expressions bordering on vulgarity and obscenity which they ordinarily would not have used. Other patients made love to persons in the room unashamedly. One woman in this series of patients indulged in love-making with a male who happened to be present even though her husband was in the room. On one occasion after indulging in a kiss, she turned to her husband and said, "You don't mind if I do this to the people that are helping us, do you?" Several patients complained that they didn't like the treatments because during this stage, they felt as if they had lost their inhibitions and they were afraid they were going to do something that they shouldn't. One young lady, who, during her illness, became dimly aware of sex desire toward her father said that her physician reminded her of her father and that she was always frightened when she awoke from a treatment because she felt that her physician had just had sexual relations with her.

There is still another type of fear reaction which came on several hours after the treatment. These patients said, "I am so afraid I will not take any more treatments. Something happened while I was waking up from the last treatment which frightened me terribly. I don't know what it was but it was so horrible I won't take any more treatments." Patients who experienced this type of fear were patients who had sex reactions characteristic of the opposite sex or sham rage awakening reactions. Other patients went from the second stage into complete consciousness and orientation so quickly that the third stage was not perceptible.

The fourth stage of the awakening reaction is *the stage of normal* (for that particular patient) *mental functioning*. We considered that the fourth stage had begun when the patient was oriented as to time, place, person and situation and showed the same restraint in regard to impulsive behavior as was exhibited prior to the treatment. Of course, many psychotic patients could not meet these requirements and did not enter the fourth stage. Many patients, after receiving several treatments, never did pass from the third stage to the fourth stage between treatments. It was commonly observed that patients who attempted to dress during the fourth stage of the awakening reaction had a tendency to fumble with zippers, had difficulty in tying shoelaces and frequently buttoned shirts or vests improperly. Several patients reported that during this stage they felt as if they were walking off a porch. These impairments resemble those described by Fulton[4] as being due to inadequate functioning of the sensori-motor cortex.

In general, patients were unlikely to have somatic awakening reactions following the first treatment but there were usually enough indications of things to come so that an experienced observer could predict whether or not awakening reactions would follow later treatments, and also what kind of awakening reactions to expect. Usually after the second, third and fourth treatments, the awakening reactions became more severe and continued so until there was clinical improvement. As the patients began to improve clinically, the severity of the awakening reactions decreased and when the patient was completely well, he was likely to sleep through the first three stages of the awakening reaction and often slept curled up in a fetal position.

One, and perhaps two, of the patients in this series exhibited what we shall call a delayed awakening reaction. The one patient who definitely had a delayed awakening reaction was being treated for an agitated depression. During the second stage, she usually was restless and performed purposeless

movements, then she turned on her side and slept for fifteen or twenty minutes. Following her fifth treatment, she had dressed and was walking down the corridor with her husband preparatory to leaving the hospital when she suddenly ran into a vacant room, kicked off her shoes and pulled off her hat, jumped up on top of a bed and began to scream and pound the wall. This disturbed behavior continued for about ten minutes and then suddenly ceased. Following this outburst, she had no memory whatsoever of what had happened and except for being somewhat out of breath and disheveled, she showed no other adverse effects from the experience. The other patient was a man with severe cerebral arteriosclerosis who had partially recovered from a hemiplegia but who was so suicidally depressed that he was given electroconvulsive therapy in spite of the risk involved. After the fifth treatment, he seemed to be much improved clinically and apparently was in the fourth stage of the awakening reaction. He was sitting on the side of the bed, smoking a cigarette and talking to his wife when suddenly, about thirty minutes after the treatment, he collapsed on the bed, stopped breathing and in spite of all efforts to resuscitate him, he expired. Autopsy failed to reveal the immediate cause of death and we can only speculate that perhaps he had some sort of delayed reaction which his lack of cardiac reserve prevented him from surviving.

Discussion

This is a study of 162 patients given a total of 1,590 electric shock treatments in which we tried to use only enough current to make certain that the patient would be rendered unconscious instantly. It is believed that most of the patients were given just enough current or only slightly more current than was required to produce a major convulsion. The motor phenomena observed in such patients were characteristic of those seen in decorticate rigidity. Some patients had what seemed to be excessively strong convulsions and when subsequent treatments were administered, the current was reduced and the patients were still rendered unconscious instantly. During the convulsions, which seemed to be excessively severe, there was spontaneous dorsiflexion of the great toes and fanning of the other toes, extension of the forearms and apneustic breathing. These observations suggest that the convulsions produced by electroconvulsive therapy are of the nature of decorticate or decerebrate rigidity rather than being due to stimulation of the cortical motor area. In this connection, it is of interest that Ward and Clark[14] were able to induce convulsions in cats by electrical stimulation of what remained of the cortex after the motor areas had been removed and Pike and Elsberg[15] found that removal of the cortical motor area bilaterally caused the convulsions produced by absinth to become tonic with no clonus.

The belief that the electric current inhibits or stops the functioning of cortical brain cells is further strengthened by the observation that immediately after the convulsion, many patients were in a state in which all that is called the personality had been extinguished. While the patients were in this state, there was observed no evidence of any functioning at a level higher than those centers in the medulla oblongata which control respiration and cardiovascular activities. As the patients recovered from the effects of the treatment, one of the first activities was that of sucking and swallowing. The center for the control of these activities is said to be in the medulla oblongata.

Just before awareness of self was re-established, many patients engaged in instinctual activities accompanied by strong emotions and this suggested that functioning had returned to the hypothalamus and its connections with the endocrine glands. Movements were deftly executed at this stage. Following the activities which suggested the return of hypothalamic functioning, the patients became aware of self and of how they felt and were able to perceive sensations. This was interpreted as meaning a return of functioning in the thalamus. The next series of activities were also instinctual and were executed with only slight evidence of super-ego restraint. Movements during this stage were not deftly executed suggesting, that the sensorimotor cortex was beginning to function but imperfectly.

The last functions to return were memory,

the ability to associate ideas, the restraints of the inhibitions acquired in our culture, and the ability to be unhappy, anxious, or miserable in connection with the symptoms of their illness. All of these functions are believed to be cortical. We have observed all gradations of unconsciousness and consciousness, from complete extinction of the personality to complete orientation associated with alertness and an ability to associate ideas and to anticipate the future. Patients seemed to be completely unconscious when they were functioning at the level of the medulla which gave way by stages to complete consciousness as hypothalamic, thalamic and cortical levels regained their ability to function. The last functions to return were associated with fine discriminations which are said by Fulton to be dependent upon the integrity of the sensorimotor cortex.

One, and perhaps two, instances of delayed reactions were observed which were accompanied by so much evidence of emotion that it was concluded that they were due to sudden firing of hypothalamic autonomic centers or perhaps of the rhinencephalon.[12]

During the stages of return of functioning following a treatment the levels of functioning described by Jackson[13] frequently manifested themselves. Not only did the levels of functioning reveal themselves but patients re-enacted their growth from the sucking infant stage to their actual chronological ages and during this process there is evidence that action patterns and memories remain intact in the nervous system even though they may be covered over in life.

The final conclusions to be drawn from these observations will be published in a later paper after reports have been made upon observations pertaining to autonomic reactions, the effects of certain drugs upon somatic and autonomic reactions and a report of diagnostic categories, number of treatments and clinical results. However, at this point we are inclined to agree with Hemphill[7] that there is a relationship between the motor phenomena during the awakening reaction and features of the patient's personality. We further conclude that the sort of person an individual happens to be, is as significant as the character of the disease with which the patient is afflicted, in determining the nature and course of the individual's illness.

Author's Note: This study was made possible by a grant from the Duke University Research Council and by the cooperation of numerous individuals on the staff of Duke Hospital and Duke University School of Medicine, Durham, N. C.

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Psychiatric Supplementation of the Medical History and Physical Examination

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Despite certain recent and invaluable contributions (see references), the literature of medicine and psychiatry still seems lacking in a concise set of reminders and directions for practitioners to follow in so supplementing their examinations as to give proper weight to factors of personality, adaptation, and "psychosomatic" relationships in general clinical practice. The following outlines, developed and used at Northwestern University, are submitted to fill this need.

Introductory Principles

Every medical disorder alters the general personal and social behavior of a patient, and this in turn affects the symptomatology, course and therapy of his illness. Similarly, environmental stresses, in persons born or made susceptible to them, may cause physiologic dysfunctions which appear as clinical symptoms and which, if severe and prolonged, may also lead to bodily disease. Therefore no medical diagnosis, prognosis or therapy is complete unless it includes an evaluation of (1) the patient's *total* deviations in behavior as well as in organic function (present illness), (2) his constitutional and characterologic development (personality tendencies) and (3) the prospects of his responding favorably or unfavorably not only to medical and surgical treatment but to interpersonal and environmental re-adaptations (prognosis and psychotherapy).

Attitude of the Psychiatrically Trained Medical Examiner:

The physician will remember, from his training and personal experiences, that under special life stresses he himself has occasionally reacted with excessive anxieties, unfounded fears, minor depressions and other deviations in thinking and mood with their accompanying physiologic expressions. Empathy so engendered will contribute to professional tact and understanding, sharpen the examiner's sensitivity to important psychiatric data, foster better doctor-patient relationships and thus, even in the first interview, lay the groundwork for effective therapy. Special interest and practice in the psychiatric aspects of the medical diagnosis will eventually make it easier for the physician to progress more directly to the essentials of the individual case, but until such skill is attained it is good practice to proceed in a thorough and orderly fashion in general accordance with this outline.

Objectives:

As indicated, it is the purpose of the psychiatric examination to elicit the following data:

I. *The present illness,* comprising the patient's complaints and the settings which precipitate, exacerbate or relieve his accompanying symptomatology.

II. *The personality evaluation,* as derived from an historical survey of the patient's *characteristic patterns* during his early childhood, in his schooling, and in his sexual, social, occupational and other relationships. The nature of previous maladaptations to environmental stress and frustration or to bodily disease are to be noted, especially with regard to the circumstances that precipitated and complicated the present illness.

III. *The mental status,* as deduced from his directly observed spontaneous behavior, and his reactions under psychological and physical examination.

IV. *Dynamic formulation:* Valid deductions from the anamnestic data as to (a) the patient's physical and intellectual endowment (b) his acquired tendencies in behavior, including special capacities and vulnerabilities and (c) the weight of other factors relevant to prognosis and therapy.

The clinical methods to be employed in

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securing this information must be varied to suit each patient, but the following general procedure will be found applicable to the great majority of patients.

I. The Present Illness

It must be remembered that in nearly all instances the patient who consults a general physician believes himself to be physically ill and will initially resent any implications that his symptoms have other than a purely organic causation. It is often advisable, therefore, to complete the usual systemic history and physical and laboratory procedures not only as an essential preliminary and differential diagnostic step, but as an indispensable basis for the further investigation of psychiatric factors. However, since a diagnosis of neurosis or psychosis can never be made by exclusion but must be based on positive data, it is well to look for such evidence fairly early in the diagnostic survey. Thus, with the medical findings reasonably complete, the patient may be tactfully but objectively informed that, whereas there is as yet little or no indication of bodily disease, his symptoms could also spring from other causes that required thorough consideration. Relatively neutral and acceptable terms suitable to the individual case (e.g., "overwork," "prolonged worry," or "emotional strain," etc.) may be employed in these introductory explanations, but it is best to avoid confusing or accusatory phrases such as "neurasthenia," "exhaustion," "psychosomatic disorder," "nervous breakdown," or "mental illness." Most patients, if they are thus made to feel that the physician is understanding and sympathetic, will more readily accept the possibility of so-called "functional" or "emotional" elements in their illness, after which their examination may be conducted as here outlined:

A. *Review of the Presenting Complaints:* Often these will in themselves be so characteristic of various psychiatric syndromes that a presumptive diagnosis can be made on this basis alone. The following are especially frequent and significant: (a) *Anxiety states:* recurrent episodes of precordial palpitation, shallow or catchy respiration, pounding pulse, sweating, tremulousness and intense subjective apprehension, with or without a feeling of a lump or obstruction in the throat (globus hystericus), urinary urgency, splanchnic constriction ("butterflies in the stomach"), giddiness or similar symptoms. Patients not infrequently complain of these as "heart attacks," "fainting spells," etc., but in most cases the symptoms are unrelated to exertion or other physical stress, and so obviously precipitated or relieved by emotional circumstances that the differential diagnosis need not be difficult. (b) *Depressive syndromes:* with typical symptoms of insomnia, anorexia or occasional bulimia, variable constipation, easy fatigueability, impotence in males and dysmenorrhea in women and variable subjective complaints of inability to concentrate, discouragement, hopelessness, hypochondriacal preoccupations, fixed melancholy, or in severe cases, thoughts of suicide. Intercurrent episodes of forced cheerfulness and distracting hyperactivity may or may not be truly hypomanic; in most instances, there is little true euphoria, and such reactions merely represent attempts to deny and overcompensate for the anxiety and depression. (c) *Phobic-obsessive-compulsive syndromes;* characterized by fears partially recognized as irrational, unwelcome but insistent and recurrent thoughts or fantasies, and ritualized behavior patterns which, when prevented or frustrated, are replaced by feelings of intense anxiety. (d) *Epileptic equivalents,* even in the absence of grand or petit mal convulsions may be suspected on the basis of episodes of clouded thinking, momentary disorientations and amnesias ("absences") or transient fugue-like automatisms, with or without post-dromal fatigue and headache. (e) *Toxic-delirious states* due to drugs (including alcohol and narcotics) or intercurrent infections may be surmised from confusions, rapidly changing hallucinatory experiences, ataxias and other motor disturbances and distorted recall. (f) *Psychotic admixtures* are revealed by bizarre ideation, gross illogicality, extreme and protracted deviations in mood or seriously altered rapport, or pathognomonic symptoms such as persistent hallucinations, grandiose and paranoid delusions and markedly impulsive behavior. (g) *Impairments of cortical func-*

tion, indicated by variable loss of memory, (especially recent integration and response, inadaptability to new or unusual tasks, and progressive deterioration of personal and social habits.

Techniques of Inquiry:

There are of course, many other psychiatrically significant configurations; the above are presented only as a reminder of the fairly common syndromes that can be revealed by an exploration of the presenting semiology. In this exploration, however, certain precautions should be followed, e.g.:

a. *Specificity of Description.* Record the patient's complaint in his own terms, since his choice of language may be illuminating. However, it may also be misleading when, for instance, a patient complains of "headache" he may have no actual cephalgia, but may instead mean a sense of inertia, or emptiness, or oppressive feelings, or other generalized discomforts vaguely referred to his "head." Other terms favored by patients because of their obscurity are "dizziness" (ranging from vertigo and slight faintness to syncope), "noises in the head" (tinnitus or obsessive sounds to auditory hallucinations), "nervousness" (meaning anything from mild restlessness and hypersensitivity to uncontrolled furors), "sleeplessness," "irritability," "tension," etc.

b. *Sensitivities and Resistances:* However, it must be remembered that many neurotic patients, aside from a symptomatic description, may wish to avoid exact formulation of illness, so that they can not be pressed too intensely at their first interview without invoking further anxiety and resistance. For the same reason—and especially if the patient shows obvious resentment—do not insist on an immediate inquisition into confidential or guarded material such as his secret drinking, his sexual habits, his marital or parental attitudes, etc. Indications of the importance of these may be evident, but their further exploration may be postponed until the patient's confidence has been more securely won.

B. Circumstances that Excerbate or Alleviate the Presenting Complaints: Information as to symptomatology can in most cases easily be obtained merely by a receptive attitude or a few evocative questions; in fact, the number and variety as well as the nature of the complaints poured forth under such circumstances may in itself be indicative of a neurotic admixture. When, however, it is necessary to make further inquiry as to the relationships of the patient's symptoms to inner emotional stresses, greater circumspection, tact and skill are called for. Once secured, nevertheless, the following data may be highly significant:

a. Typical settings in which anxiety, phobias, obsessions, etc, are evoked: e.g. when the patient is called upon to perform an unwanted task, when he is reprimanded by some authority, or when he is otherwise rendered fearful, resentful or hostile.

b. Possible correlation for an otherwise unaccountable periodicity in the patient's symptomatology, e g.: migraine headaches which appear only on week-ends when a wife must contend with the continuous presence of her husband and children, or recurrent sexual impotence in a husband who unconsciously wishes to avoid impregnating his wife during her presumably fertile sexual cycle.

c. The nature of the symptoms may likewise be revealing, e.g.: gastro-intestinal dysfunctions in immature, dependent patients under circumstances in which they feel deprived and rejected, or urgency and diarrhea in aggressive individuals who feel suddenly frustrated and angry. However, in the absence of specific evidence, care must be taken not to make untenable presumptions about the specificity of such so-called "psycho-somatic" relationships.

d. The circumstances under which symptoms are relieved may also be psychologically significant, e.g.: change of work, vacations, moving the home, alterations in the family or social constellation, hospitalizations, and so on. Particularly significant is the history that the patient had felt initially benefited by a wide variety of treatments (including simple sedatives) administered by many doctors, but that in each case the "illness" returned as the physician's enthusiasm flagged, after which the patient would seek another with renewed complaints. This, of course, also has an important bearing on current prognosis and plans of therapy.

e. Preliminary information may likewise be gathered about the deliberate or unconscious advantages which the patient may be deriving from his illness. These may take the form not only of financial compensation and relief from responsibilities ("secondary gain") but may also satisfy security-seeking, regressive, aggressive, or other "primary" neurotic purposes.

C. Precipitation of the Present Illness: Whenever possible the physician should determine the circumstances which, operating on a previously sensitized individual (*v.i.* under Personality Development), served to precipitate his current symptoms. In the case of combat or post-catastrophic neuroses the history may be fairly obvious, but in most neuroses, though the stresses involved were taken just as seriously by the patient, they are far less evident to an outside observer. In such instances they may consist of more subtle but equally disruptive

threats such as a dreaded demotion or promotion, guilt over the secretly desired illness of a rival, an impending engagement or marriage, or various social or religious events or transgression of great symbolic significance to the patient. Inquiry into such causes must be especially skillful, but is often highly illuminating.

II. The Personal History

Since psychiatric disorders are exacerbations of previously latent behavior tendencies it follows that every neurotic or psychotic illness is an outgrowth of the patient's previous personality, and that the formation of the latter must be traced as fully as practicable from early familial influences through the vicissitudes of latter life to the onset of his current disabilities. Training and practice in this task will make it far less formidable and time-consuming than it first appears, but here as elsewhere in the psychiatric examination the physician must remember that most patients will be ignorant of, and unconsciously resist recognizing, any connection between their behavior and certain cherished attitudes and patterns derived from their past experiences. This may be particularly true when crude or even merely routine attempts are made to elicit the familial and personal history; under such circumstances the patient may well defend himself, verbally or covertly, with some rationalization such as: "I came here for relief from headaches and bowel trouble, and they send me to a mental clinic where the doctor asks me if my dear old grandmother was insane, and whether I really like my wife. Who's crazy?" Such reactions may be avoided only if the patient is given a preliminary understanding of the importance of a personal history in better diagnoses and more effective therapy, and if the interview continues to be conducted with proper empathy and adroitness. The following considerations are helpful:

a. *Interview Arrangements:* Whenever practicable, choose a quiet, preferably soundproof office free of distractions and interruptions and reassure the patient that all data will be kept professionally confidential. The examiner should be sufficiently familiar with the nature of the information he seeks to make it unnecessary to consult this or any other outline during the examination; to do so implies incompetence and impairs the ease, rapport and effectiveness of the relationship. For the same reasons it is best to avoid voluminous writing during the interview, but accurate notes should be taken of key facts and expressions for later elaboration.

b. *Transitions in Content:* If the patient has, during his account of the present illness, vaguely or euphemistically described himself as "nervous," "emotional" or "high-strung" it may later be asked if these or other characteristics were familial traits, thus leading the patient to give significant descriptions of his parents, antecedents, and siblings—e.g., the "family history." Similarly, logical transitions may be made from the present illness to various categories of the past history by enquiry as whether current or other symptoms were present in childhood, whether they were influenced by excessive study in school, "overwork" or other maladjustments at early jobs, the stresses of various social adaptations and so on. Information on particularly guarded topics such as sexuality may be more readily secured by an indirect anamnestic approach: a complaint of dysmenorrhea may be used as a basis for asking the patient to describe the circumstances of her menarche; once the subject is broached, she may more easily be led to describe her sexual fears and fantasies and possibly neurotic reactions and sexual traumata. Similarly, tracing a complaint of ejaculatio praecox in a male patient may reveal familial prohibitions of sex activity and later fears of masturbation or homosexuality, directly related to his present marital or other difficulties.

c. *"Irrelevancy."* On the other hand, the patient's tendencies to surface diffuseness or seeming irrelevancy when given an opportunity to talk spontaneously and freely—as should be done insofar as practicable—may in themselves be directly significant if basic associative themes are detected. For example, a patient may "wander" quite significantly from an account of her marital unhappiness to a description of her idyllic childhood with her widowed mother, or an aggressively competitive individual may "inadvertently" touch on his own multiple superiorities to others, including by implication the physician. With limited time, a completely undirected interview is rarely as informative as a skillfully guided one, but both techniques may be used as indicated by the experienced examiner.

d. *Sources of the Personal History:* In general, the patient himself is the most fertile and significant source for the needed data, but wherever necessary or available, other sources may be used: interviews with family and friends, perusal of physicians', employers' or social service reports, school or military records, etc. It must be remembered, however, that all such sources have various limitations and biases so that each may require individual evaluation and check against the others.

e. *Significance of the Data:* It will be recognized that a psychiatric history is not merely a chronological account of the patient's life, but an attempt to discern those *characteristic patterns of behavior* which constitute his personality and there-

fore possess psychiatric diagnostic and prognostic value. The aim, therefore, is not only to determine *what happened to* the patient but also (a) *his total responses to what happened* and (b) perhaps how, consciously or not, *he arranged for the repetition of similar experiences.* It is acknowledged that because of the patient's defenses and biases complete "accuracy" is unattainable, but even if his own accounts of past happenings are deliberately falsified or unconsciously distorted by symbolic ("screen") memories, the nature of these distortions will also be psychiatrically significant. In this sense, the proffered "history" is in itself an excellent index of the patient's mental status, in that it furnishes valuable data as to his motivations, fantasies, self-concepts, and current interpersonal relationships.

With these techniques and qualifications, the information secured under the heading of personal history may be grouped under the following categories:

1. *The Family History.* Although it is highly questionable whether there are significant hereditary factors in neurotic (or many psychotic) disorders, it is undeniable that the personality of every adult is greatly influenced by those with whom he comes into intimate contact in childhood. Therefore, even apart from its genetic significance the patient's own description of the personal characteristics of his grandparents, parents, siblings or other relatives is often significant, particularly when special aspects of, or gross deviations in, their behavior (over-indulgence, tyranny, unpredictability, tempestuousness, sexual seduction, etc.) directly affected the patient's early formative experiences. Note also his subsequent allegiances or aversions to parent-surrogates, such as governesses or teachers, attachments to and rivalries with various siblings, and the adverse influences of familial discord, divorce or desertion.

2. *Birth and Early Development:* Accurate data may be difficult or impossible to obtain, but the patient's or other informant's impressions about the following may be significant: prematurity; ease or difficulty in delivery (asphyxia, injuries); type of nursing and methods of weaning; delays in early habit acquisitions such as sitting, walking, word or sentence formations and toilet training; play interests; manifestations of early neurotic tendencies such as feeding difficulties, excessive crying, night terrors, tics, spasms, phobias, persistent thumb sucking or enuresis, etc.

3. *Schooling and Initial Socialization:* Ages of enrollment and at various grades attained; adjustment to school routine and discipline; scholastic and extra-curricular activities liked and disliked; development, nature and depth of leadership or group loyalties at various ages and unusual dependence upon or friction with teachers or other school authorities. Repeated disciplinary difficulties, social isolations, truancies and delinquencies are especially significant.

4. *Sexual Development and Attitudes:* Tactful, objective inquiry into early sex preoccupations (autoerotisms, curiosities, phantasies, fears, inhibitions, displacements); later development, nature pace and intensity of homo- or heterosexual expressions; deviations in technique or unusual attachments or aversions to sex partners. Previous and current marital adjustments including not only sexual compatibility but also relative fidelity or promiscuity, use and justification of contraceptive techniques, attitudes to in-laws and children, mutual interests and other interpersonal conflicts and satisfactions. In most cases, it will be found that the latter considerations determine sexual adjustments in marriage, rather than vice versa.

5. *Other Habit Developments:* concerning food, sleep, dress, hobbies, recreations, etc. In later life the survey should include habitual overindulgence in tobacco, caffein drinks, alcohol and sedative or stimulant drugs.

6. *General Social Adjustments:* Type and constancies of friendships and group loyalties; submissiveness or leadership in joint activities. Nature and intensity of significant social, ethical and religious sentiments, with special relation to unusual rigidities or prejudices. Circumstances leading to compatibility and belongingness or, conversely, feelings of diffidence, embarrassment, resentment and social failure.

7. *Occupational History:* Types of work sought or idealized; jobs secured and periods held; variability of effort and application; acceptance of duties and cooperation, or competitiveness with fellow-workers; reactions to supposed injustice, idleness and failures, or to promotions and successes; evaluation of employers, superiors or employees.

8. *Military Service:* Dates; branch; reactions to training, discipline and combat; demotions and promotions; motivations at discharge; pensions sought and retained; current attitudes.

9. *Effects of Injuries and Illnesses:* An account of the patient's previous organic illnesses, including a description of significant reactions to them such as excessive invalidism, prolonged disability and dependency or lack of cooperation with medical care. Accident-proneness and consequent litigation may also be significant. Note all hospitalizations.

10. *Transition to Present Illness:* Under this heading the psychiatric anamnesis may be completed by a consideration of the circumstances that, singly or in combination as outlined above, interacted with the special sensitivities and vulnerabilities of the patient to precipitate his present illness; and later to seek (or be referred for) psychiatric help. Not infrequently, however, a complete demarcation cannot really be made, and the "present illness" will now be seen as merely a continuation, or at most an exacerbation, of the patient's previous life patterns and neurotic tendencies.

III. The Current ("Mental") Status

A. *Direct Observation:* In most previous guides for psychiatric history taking a great deal of emphasis was placed on this portion of the psychiatric examination, since it was

intended to reveal gross deviations in motivation, affect, thinking and behavior relevant to the differential diagnosis of various forms of "mental deficiency" and organic or "major" psychoses. The medical and surgical clinician must, of course, remain alert to these possibilities; however, a far larger number of his patients will have only characterologic, neurotic or functional somatic disturbances, and in these direct and crude investigations of intellectual capacities, disorientations or hallucinatory experiences may be not only unnecessary but needlessly disturbing and alienating. Actually, valid information as to most of the rubrics of the so-called "mental status" will already have been gathered during the course of the history and physical examination, as will be indicated by the following review:

1. *General Behavior:* As manifested by the patients appearance, gait, posture, demeanor, dress and personal hygiene, mannerisms, tics, characteristic activity, obvious idiosyncracies or compulsions, cooperation with office or clinic procedures, and general responses to the examiner and others.

2. *Intellectual Level:* This may be deduced fairly accurately from the quickness and accuracy of the patient's perceptions, the versatility and specificity of his vocabulary, his capacities for both concrete and abstract thinking, the scope and accuracy of his general information, the richness of his imagery, his forms of wit and humor and the general nature of his cultural attainments. In estimating basic intellectual capacities, however, allowances must be made for the previous presence or absence of social and educational opportunities.

3. *Sensorium:* Memory, retention, associative and communicative capacities, orientation, etc., are not organically impaired in the neuroses. If, however, intellectual failures possibly due to cerebral dysrhythmia, trauma or disease are indicated by gross discrepancies in dates, places and relationships in the history, the patient's sensorium may be further checked by indirect questions designed to reveal his awareness of identity, time and locale, his capacity to remember his past accurately, to repeat forward or backward a series of from four to eight numbers given to him, to subtract 7 serially from 100 (ostensibly as a test of concentration), to retell an anecdote, to act on a set of simple directions adequately, and so on. Even when necessary, this portion of the examination may be so conducted that the patient need not resent or hardly be aware of the fact that he is being tested.

4. *Mood and Emotional State:* The fixity or range of the patient's feelings and affects: e.g., anxiety, depression, indifference, euphoria, hostility, etc., will have been in part verbally communicated by him during the interview and examination; in addition, the examiner may have noted such physiologic and mimetic accompaniments as flushings, lachrymation, pupillary dilations, tremors, jaw-clenchings and various muscular tensions, sudden startle reactions, and other visceral and motor expressions of emotion. The stooped posture, slow movements, low retarded speech and dolorous facies of the melancholia are especially noticeable; in such patients, the possibility of self-destructive preoccupations may be better investigated gently and indirectly (e.g. "Are you sometimes so discouraged about your illness that you feel there is no use in going on?") than by direct questions as to frank plans for suicide. With empathy and experience the examiner will also more readily sense the touchy apprehensiveness of the anxiety neurotic, the bland dependency of the so-called "conversion hysteric," the strained, restless, pseudoeuphoria of the hypomanic, the demanding, self-pitying, depressive and the dereistic withdrawal and impaired or deviant rapport of the schizoid or schizophrenic.

5. *Preoccupations and Special Symptoms:* When indicated, further inquiry may reveal persistent fantasies or significant dreams or nightmares, or phobias, obsessive preoccupations, compulsions, hypochondriacal tendencies or fixations, diurnal or other variations in energy-level or temperament, recurrent déjà-vu experiences, feelings of depersonalization, confusion or panic, transient absences or prolonged fugue states, etc. Even more serious psychological deviations may incidentally be uncovered: for instance, hallucinations may be described in response to an inquiry about "very vivid daydreams or imaginations," whereas paranoid or other delusions may emerge clearly if they are detected and accepted as possibilities (though never expressly agreed to) during the interview.

6. *Insight:* This is always a complex, relative and variable aspect of the patient's past attitudes and current status, but from a clinical standpoint insight may be estimated along the following dimensions: (a) the patient's own explanations of his illness, (b) his ideas as to prognosis and treatment and (c) the sincerity of his desires for, and cooperation with, medical and psychiatric therapy. The mere agreement of the patient with the examiners' formulations should not ipso facto be termed "insight."

B. *Psychiatric Significance of the Physical Examination.*

The patient's conduct during this procedure often contributes evidence as to his mental status: e.g.: excessive shyness, modesty, or exhibitionism; extreme passivity; suspiciousness or hypersensitiveness with regard to abdominal, vaginal or rectal examinations; irrational fear of transillumination or retinoscopy, and so on. Incidental observations should include the adequacy of oral, ungual, and other personal hygiene,

tatoo marks, drug rashes, evidences of minor scarrings from repeated syncope, or signs of hypodermic or intravenous use of narcotics.

C. *Supplementary Psychologic Testing:*

Whenever there are difficulties in differential diagnoses or other special problems the patient may be referred to a trained clinical psychologist for the following:

(a) Tests of intellectual functions, such as the Wechsler-Bellevue or modified Stanford (b) Tests of occupational preference or special abilities, such as the Strong Vocational Guidance or the Seashore scales of musical aptitude. (c) Personality tests, of which the questionnaire techniques such as the Bernreuter or the Neymann-Kohlstedt are least reliable, and the projective techniques such as the Rorschach and the Thematic Apperception are the most revealing.

D. *Ancillary Techniques in Psychiatric Diagnosis:*

These include polygraphic recordings during questioning or history taking ("lie detection"), hypnotic catharsis or regression, or the administration of drugs such as scopolamine ("truth serum") or intravenous sodium pentathol or sodium amytal. Such procedures are rarely if ever necessary in psychiatric diagnosis, carry definite physical and psychologic dangers and had best be left to the specialist; here too, however, the physician should be acquainted with their general significance.

IV. Psychiatric Formulation

When sufficient information as outlined above has been gathered to make possible a comprehensive formulation, this should be integrated and summarized according to the following outline:

1. Brief statement of the precipitating circumstances, nature, duration, and course of the present illness.
2. Organic factors as revealed by the medical history and by the physical, neurological, medical and other special examinations.
3. Constitutional and personality factors as indicated by the familial and personal history.
4. The current behavioral status as shown by direct psychiatric observation and supplementary psychologic or other tests.
5. An interim diagnostic summary based on the above, including: (a) the presence and relative prominence of various psychiatric syndromes (e.g., anxiety, depression, organ neuroses, schizoid tendencies, etc.)

(b) the relationship of these reactions to specific external stresses (environmental maladaptations) and (c) their roots in inner motivations, formative experiences and derived character patterns (psychodynamics).

6. Indications for further diagnostic procedures, medical or psychiatric.
7. Preliminary plans for therapy, as recorded under the following rubrics:

a. Immediate symptomatic relief by medication, sedation, physiotherapy, or other medical or surgical procedures. b. Environmental alterations such as change of residence or work, or environment, or, if necessary, hospitalization for total care. c. Projected nature and techniques of the psychotherapy to be employed. d. Methods of securing the cooperation of the patient's friends and family in his treatment or, when necessary in his legal or custodial care.

This formulation should be supplemented by progress notes as the case is treated and followed, and elaborated and amended as new facts become available.

The above represents a fairly extended but by no means all-inclusive review of modern approaches to a comprehensive and dynamically oriented clinical examination. As a summary, the following Condensed Guide may be employed for ready reference in actual clinical usage.

Condensed Outline for the Psychiatric Supplementation of the Medical Examination

Rationale: Since the symptomatology, course and therapy of every illness is influenced by the motivations, personality and environment of the patient, no medical diagnosis or plan of therapy is complete without an adequate survey of these factors. With proper sympathy and tact therefore, the examining physician should obtain the following information:

I. *Psychiatric Aspects of the Present Illness:*

1. *Neurotic Symptomatology:* Determine the intensity and frequency of anxiety reactions, obsessive-phobic-compulsive states, hypomanic or depressive mood swings, disturbances of consciousness or motor control, drug intake, and recent impairments of intellectual functions and interpretations of reality.

2. *Relationship to Environmental Stresses:* Survey the circumstances that precipitated, exacerbated or alleviated the above deviations in behavior and their accompanying organic (psychosomatic) dysfunctions, including the patient's reactions to previous examinations and therapy.

II. *Personal History:* Since normal and abnormal behavior can be best understood as an evolution of genetic, constitutional and adaptational patterns in relation to physical and emotional stresses, this section of the examination should comprise a pertinent review of the following data about the patient as a person:

1. *The Family History:* Brief descriptions of significant traits in parents, siblings or important relatives.

2. *Birth and Early Development:* Data on infantile traumata or illnesses, feeding disturbances, spasms, terrors, or other childhood neurotic tendencies.

3. *Schooling and Early Socialization:* Scholastic interests and performance, adjustments to discipline and group participations.

4. *Sexual Attitudes:* Including marked deviations, special fears, and marital and parental adjustments.

5. *Habits:* Of food, sleep, recreation, and intake of alcohol, sedatives or other drugs.

6. *Social Adjustments:* Friendships; social belongingness; recreations; religious activities.

7. *Occupational:* Goals; level of initiative and performance; cooperation with employers, fellows and subordinates.

8. *Military Service:* Motivation and reactions in training or combat.

9. *Previous Reactions to Injuries or Illnesses,* including tendencies to emotional over-reactions, dependency and invalidism.

The personal history is less a chronological account than a biographical review of the *origin and nature of the patient's characteristic patterns of conduct,* and as such will lead naturally into the precipitation of the present illness as in I_2 above.

III. *The Current ("Mental") Status.* This consists of a summary of the patient's behavior as either directly observed during history-taking and physical examination, or as elicited by further inquiry. The data may be organized under the following headings:

1. *General Behavior:* Appearance, posture, mannerisms, dress, personal hygiene, etc.

2. *Intellectual Level,* as estimated from perceptive grasp, vocabulary, general information, logic and versatility of thought and cultural attainments.

3. *Sensorium:* Orientations; to time, space and identity; clarity of awareness and retention.

4. *Mood and Emotional State:* Excessive anxiety, euphoria, passivity, hostility, indifference, etc. In melancholic patients the possibility of suicidal impulses is of immediate practical importance.

5. *Special Symptoms,* such as hypochondriacal preoccupations, panic or fugue states; déjà-vu, clouded consciousness, etc.

6. *Psychotic Manifestations:* Affective distortions, hallucinations, grandiose or persecutory delusions and paranoid systematizations are seen relatively rarely in general clinical practice, but must be recognized when they occur.

7. *Insight:* The patient's own concepts as to (a) the nature of his illness, (b) his ideas as to treatment and prognosis, (c) his cooperation with examination and therapy.

IV. *Formulation.* The above information may be integrated and appended to the medical summary under the following headings:

1. *Organic Factors* in the present illness, as determined by the medical history and physical and laboratory findings.

2. *Psychiatric and "Psychosomatic" Components* of the current illness, and their predisposing and precipitating circumstances as determined by the psychiatric and personal history and mental status.

3. *Indications for Further Diagnostic Procedures;* medical, psychologic or special techniques.

4. *Psychiatric Diagnoses and Prognosis.*

5. *Treatment:* Comprising methods of immediate systematic relief or environmental readjustments, need of hospitalization for special procedures, and plans for long-term medical and psychiatric treatment and eventual social rehabilitation.

With sufficient familiarity and skill these important psychiatric aspects of the medical work-up need take relatively little time, and will add immeasurably to comprehensive diagnoses and effective therapy.

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Control of the Depth of Barbiturate Anesthesia by Means of Low Amperage Currents

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Barbiturate anesthesia is being used in increasing frequency in non-convulsive shock therapy in the treatment of mental illness.[1,2] In this form of therapy, it is desired that the patient undergo the treatment smoothly and without pain during the entire four minutes of its administration. This can only be accomplished with anesthesia of adequate depth; for without it, the current is very painful, and it becomes impossible to give the treatment. The ordinary patient can tolerate only ¼ milliampere of current without anesthesia, whereas up to 10 milliamperes or more are necessary for adequate non-convulsive therapy. The usual methods for testing for the depth of anesthesia by the corneal reflex, relaxation of the jaw, lid reflex, yawning, etc., are unreliable and variable, resulting sometime in overdosage, but more frequently in under-dosage which precludes the administration of the non-convulsive shock treatment. The practice of injecting all patients with a fixed dosage of a barbiturate is unscientific and fraught with danger from overdosage and possible fatality.

We have devised a simple test, which makes it possible to obtain the correct depth of anesthesia for non-convulsive shock therapy:

Test: The machine with which the treatment is to be given is used for the test. We have been using a Reiter CW47 which delivers a spiked unidirectional current ranging from 0 to 20 milliamperes. The current selector knob is turned to position one. The two electrodes are placed either over the temples or in the parietal regions, an inch above the ears, and connected to the machines. The needle is introduced in a suitable vein and the patient is engaged in conversation or made to count. The barbiturate solution—we have been using 5% thiopental sodium (sodium pentothal (R))—is administered slowly at the rate of one cc. (50 mg.) each 20 seconds until speech becomes slurred or the patient begins to miss the count. At this point, the injection is stopped, the needle is not withdrawn and 30 seconds are allowed to elapse for the drug to develop its full action. The current is then turned on. Beginning at zero milliamperes, it is slowly increased, at the same time carefully observing the patient for any signs of pain, which he manifests either by protesting, grimacing or moving some part of his body. If this occurs before the meter registers 2½ milliamperes, the patient is not as yet adequately anesthetized. The current is turned back to zero, another one cc. of pentothal solution is given, and the procedure is repeated. When the patient is able to tolerate 2½ milliamperes of current without pain, he is adequately anesthetized and will tolerate up to 10 milliamperes or more comfortably.

We have used this test hundreds of times during the past two years and have found it reliable and useful. On a number of occasions, as in a patient who became adequately anesthetized with only 50 mg. of pentothal, the test has saved us from serious difficulty. We have found that from one cc. (50 mg.) to 12 cc. (600 mg.), or rarely more, of sodium pentothal are required for adequate anesthesia in non-convulsive therapy. We can recommend this test to determine the depth of anesthesia in non-convulsive shock therapy. It may possibly be of use in other types of anesthesia.

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Trichinosis as a Cause of Encephalitis

(Case Report and Review of the Literature)

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Trichinosis of the central nervous system, not unlike CNS lues, is capable of simulating a number of other diseases, namely encephalomyelitis, polyneuritis, acute anterior poliomyelitis, meningitis, dermatomyositis, periarteritis nodosa, and even psychosis.[1] In reviewing the literature, we very rarely found two cases of CNS trichinosis in which the signs, symptoms, and laboratory data were alike. From this observation and from the experience of our own case, we noted that when the trichinella spiralis invades the central nervous system, it has no predilection for any particular part of that system. This accounts in large measure for the frequency of incorrect diagnoses, especially in the early stages, and for this reason, we feel that a report of our study should be interesting and valuable.

Case History

F.F., a 33 year old, white, divorced, female was admitted to New York City Hospital with a provisional diagnosis of poliomyelitis or encephalitis due to trichinosis made by her personal physician. The chief complaints were severe headache, blurred vision, inability to walk, and a numb feeling over the entire right side of the body including arm and leg.

About one week prior to admission, she noticed a puffy appearance around both eyes and a numb feeling over the entire left side of the body. On this basis plus a blood count and a history of frequenting Chinese restaurants for pork dishes, a diagnosis of encephalitis was made. At no time during the early days of the disease were there any signs of pyrexia, diarrhea, vomiting, anorexia, or muscle aching, and by the time of admission, the numb feeling had shifted from the left side to the right side.

She had been treated at a private hospital for menorrhagea and metorrhagea two months prior to her present admission. At that time a diagnosis of bilateral tubo-ovarian disease and polypoid hyperplasia was made after a curretage of the uterus and a biopsy of the cervix were performed. After a blood transfusion she improved rapidly and was discharged.

Physical examination revealed a well nourished female with bleached blonde hair. Temperature was 98.8; pulse 80, strong and regular. Her head and face appeared symmetrical. Her tongue was not furred. Chest examination showed a well developed chest with clear breath sounds of good quality. In examination of the cardio-vascular system, her blood pressure was 105/75. There was no distension of the great veins of the neck and no signs of cardiac enlargement. The ventricular rate was 80. The rhythm was regular. The sounds were of good quality, and no murmurs were noted. The abdomen was supple and no palpable masses were noted. The liver and spleen were not palpable. Extremities were well developed; no varicosities were found; no rashes or skin eruptions were present; no adenopathies were noted. Pelvic examination was deferred.

Neurological Examination:

Physical Functions: Intelligence appeared good; attentiveness was fair; memory appeared good; she was well oriented as to time, place, and person. She was emotionally upset about her inability to walk, and several outbursts of crying occurred. She was depressed and mentioned that she had other troubles and worries besides her present illness. There was no evidence of hallucinations or delusions.

Speech and Articulation: The patient was right-handed. There was no aphasia or apraxia and articulation was perfect.

Cranial Nerves: Examination for smell was deferred. Visual acuity was diminished as the patient was unable to read a news-

From the Neurological Service of City Hospital, Welfare Island, N. Y. C.

paper at any distance. Visual fields were not plotted at this time. The optic discs and fundi appeared normal. External ocular movements were normal and no conjugate deviation was noted. Both pupils were equal and reacted to both accommodation and light. Both corneal reflexes were noted. Sensation over the face was normal. Facial movements were normal, and the face appeared symmetrical. Hearing was grossly normal bilaterally. The palate was symmetrical and not hanging; the uvula was in the mid-line. The tongue showed no signs of deviation. The sterno-cleido-mastoid and trapezius muscles appeared normal bilaterally.

Motor Functions: Power appeared greatly diminished in her right arm and leg. Coordination was good and no intention tremor was noted. Muscles tonus appeared good. There were no signs of wasting. No fibrillation was seen, nor were there any signs of hypertrophy. There were no involuntary movements or seizures.

Cutaneous Functions: Touch as tested by cotton was normal and pain as tested by pin-prick was normal.

Deep Sensibility: Joint sense and vibratory sense appeared normal in all respects. Sensibility of muscles and tendons to deep pressure was normal and no muscle tenderness was observed on palpation. Tactile localization appeared normal.

Reflex Functions: Deep tendon reflexes appeared more active on the right. The left ankle jerk was absent. No pateller or ankle clonus was present. The epigastric reflexes were present as were the upper and lower abdominal reflexes. No Babinski was present. There was no spincteric impairment. Kernig's sign and Brudzinski's sign were both negative. The spine and cranium showed no deformities or tenderness and no decubiti or other perforating ulcers were present.

Since there was no concrete evidence of trichinosis in the history or physical examination, the very first impression was that the patient might be suffering from an intoxication of the central nervous system of some unknown etiology with perhaps an element of hysteria present.

Course and Laboratory Data

The morning after admission, an examination of the blood revealed an hematocrit of 37, hemoglobin of 10.7 grams or 78%. The total white cell count was 11,800. The differential count revealed 74% of polymorphs, 15% of lymphocytes, 9% of eosinophiles, and 2% of basophiles. The blood Wassermann was negative. A lumbar puncture was done, and the initial pressure was equal to 120 mm of water; Pandy was negative; 103 cells were counted, two of which appeared to be eosinophiles and the majority polymorphs. Chemical analysis of the fluid revealed 68 mg % of protein and 0.033 mg % of sugar. The urine was negative in every respect.

Two days after admission, the patient's evening temperature reached 101.8 degrees. Her pulse was 100 beats per minute, regular, full, and strong. Respirations were 24 per minute. An examination of the heart and chest revealed no abnormalities of these structures other than the tachycardia. Her neurological status remained the same. Her chief complaints still were inability to walk, blurred vision, and severe headaches. She was put on 300,000 units of procaine penicillin daily and was given aspirin for headache and discomfort.

By the following morning her temperature had dropped to 98.6 degrees, but in the evening, it rose to 100. After that, her temperature fluctuated between 98.6 and 100 degrees until the fourteenth day after her admission. From that time on, it remained within normal limits for the remainder of her stay in the hospital.

Even on the fourth day, we were not convinced that we were dealing with a case of trichinosis since there weren't any muscle aches or gastro-intestinal signs or symptoms. As for the eosinophilia of 9%, we were aware that numerous other conditions might be responsible for a rise in eosinophiles, namely: dermatitis herpetiformis prurigo, pemphigus, psoriasis, asthma or other allergic states, in members of an allergic family who may not themselves have manifested the allergy, in lymphadenoma especially when the liver is involved, after tuberculin injections, during recovery from

infections (post-infective or post-toxic eosinophilia), during the absorption of an hemorrhagic pleural effusion, quite often in myeloid leukemia, occasionally in polycythemia vera, and after taking certain drugs i.e. Nirvanol.[2] Other possibilities considered were atypical polioencephalitis and periarteritis nodosa.

As a precautionary measure the Health Department was notified and several consultations were held with their consultants. On the subject of diagnosis, they all shared our views. They suggested isolation and a diagnosis of an atypical encephalomyelitis of unknown etiology until a more definite diagnosis could be established.

On the fifth day, a stool examination for parasites was negative. In vaginal examination there was a lemon colored discharge. A microscopic examination of this material showed that it contained many pus cells and thin gram positive bacilla but no gram negative diplococca. A rectal examination revealed no abnormal findings. A spinal tap was repeated and the pressure found to be within normal limits. About 80 cells were counted in a space of one square mm. Total protein was 0.044 mg. %. Kolmer was negative, and the colloidal gold curve read: 0000000000. A compliment-fixation test for trichinosis performed at the Health Department laboratory was positive. However, this department specifically stated that a positive test does not necessarily indicate that the individual is suffering from clinical trichinosis at the time the serum was obtained since non-specific reactions may be encountered and that periarteritis will usually give a false positive reaction in this test.

Aspirin was still prescribed for her headaches and discomfort and at times it was necessary to add codeine. Terramycin 250 mg was ordered every six hours. In addition, a "City Hospital Cocktail" consisting of 200 mg of thiamine chloride, 300 mg of nicotinic acid, and 500 mg of ascorbic acid prepared in one ampule of calcium gluconate was given intravenously every other day as a general tonic. Two cc of crude liver extract was also added to this regime to be given intramuscularly twice a week.

On the eleventh day temperature, pulse, and respiration were normal. She appeared quite euphoric, as was noted for the past two days. She claimed her vision was improving and that she was now able to read larger print. An ophthalmology consultation revealed no ocular pathology. There was still some hyper-reflexia of the upper and lower right extremities. A right Babinski was noted which seemed to be transient in nature. For the first time, intention tremor was noted on picking up a coin with the right hand, on placing the right index finger on the nose, and on placing the right heel on the left knee. Dysdiadokokinesia of the right hand was also noted. At this time, it was thought that if polioencephalomyelitis was present, it might well be of the cerebellar type.[3] Nystagmoid movements of both eyes in the horizontal plane was also observed at this time.

On the thirteenth day she began to complain of backache and of a "drawing" sensation in the area of the left shoulder. At this point, a biopsy of the left deltoid muscle was performed and sent to the pathology laboratory for examination.

Pathology Report: Throughout the sections of striated muscle, there were focal areas of acute necrosis of muscle in which the fibers had lost their striations and showed a marked increase in nuclei. Throughout the muscle and intermuscular tissue, there was a severe inflammatory cell infiltration consisting of polymorphonuclears, lymphocytes, plasma cells, and eosinophiles. There was an area showing an encysted parasite.

Diagnosis: Trichinosis with acute and chronic myositis.

After the pathological diagnosis was established, the patient was given several injections of mepharson, but it is doubtful whether this drug had any beneficial effect on the course of the disease.

X-Ray and EKG: X-rays of the skull and chest were negative. An EKG revealed: regular sinus rhythm, PR=0.16 sec., left axis deviation, semi-horizontal heart, and no significant abnormalities.

On the twentieth day, a lumbar puncture revealed pressure within normal limits; the fluid was clear, and 20 lymphocytes were counted. No trichina larvae could be found in the spinal fluid. Another compliment-fixation test and a flocculation test for trichi-

nosis showed the former to be negative, but the latter was 3 + positive in undiluted fluid. The Kolmer was negative but this time the colloidal gold curve was: 5432100000. The total protein was 0.027 mg %.

On the twenty-seventh day she continued to have pain in the left shoulder and in addition, pain across the left side of the chest. Nystagmoid movements of her eyes were no longer noted. Intention tremor and incoordination previously noted, now appeared minimal, and the patient improved in spirits despite her discomfort.

On the twenty-eighth day she still complained of left shoulder and left chest pain. A neurological examination at this time revealed that the deep tendon reflexes on the right were more active than in the left. A Hoffman sign and Wallenberg sign were noted on the right but were absent on the left. The knee jerk on the right was hyperactive and resulted in a cerebellar pendulous movement of the right foot. There appeared to be an equivocal Babinski on the right. The patient was now able to walk, but with a cerebellar gait reeling toward the right. She was forced to walk this way she claimed, because her right knee kept "giving out." A slight degree of incoordination was still noted on placing her right index finger on her nose. The headaches, however, had entirely ceased. Since the early days of her admission, she had been receiving passive muscular exercise but now she was given active muscle exercises.

On the thirty-third day her condition was considered good, but she still complained of a tight feeling in her left shoulder and left arm. Her gait was still the same in nature but showed improvement.

On the forty-third day she was anxious to go home and continue with physio-therapy at an out-patient clinic. Hense, she was discharged with a slight residual paresis of the right arm and leg and a slight right cerebellar ataxia. Whether her abnormal neurological signs will completely vanish or not, only time will tell.

Discussion

It is evident that the inconsistency, the irregularity, and the non-conformance to pattern in which the signs and symptoms appear in trichinosis of the central nervous system is the rule rather than the exception.[1,4,5,6,7,8,9,10] We have endeavored to illustrate in our own case how CNS trichinosis was ushered in without the usual signs and symptoms of trichinosis; how at first one of the cranial nerves and the pyramidal system were attacked, these signs later disappearing only to be followed by evidence of cerebellar involvement. Signs of cerebellar and pyramidal pathology existed at the same time, and finally the cerebellar signs alone remained.

We wish to point out that the laboratory findings may vary widely. In our own case, the eosinophilia was moderate, whereas in the first case of Houston, it was 41%.[8] As a matter of fact, an elevation in the number of eosinophiles need not necessarily be present.[4,11,12] On two different spinal taps, we found two different colloidal curves, one entirely flat and the other, pareticlike, whereas in the case of Bruno and Goodgold, two different taps revealed two flat curves.[4] We always noted a clear cerebrospinal fluid, whereas in Skinner's first case the fluid was xanthochromic and contained 700 crenated cells per cc.[5]

Pathology

In order to be able to visualize the pathological process and mechanism of CNS trichinosis, a brief review of the life cycle of the trichinella spiralis and of the general disease seems at this time appropriate. Trichinosis or trichiniasis is considered to be an acute, specific fever. In a typical textbook case, the early signs and symptoms are abdominal pain, vomiting, diarrhea, and anorexia, and in one to two weeks, a rise of body temperature and then muscle aches. This aching is due to a myositis produced by the migration of the larvae of the organism. If the migrating larvae are few in number, there may not be any symptoms. On the other hand, in a severe case, death may ensue. When recovery takes place, there is an encystment of the larvae in the muscle itself. Any muscle of the body, including the diaphragm and heart can be invaded.

The infective agent is a nematode worm, and the infection is contracted from eating raw or undercooked pork. It has also been known to exist in bear meat. The hog obtains the infection by eating infected rats.

When the contaminated meat harboring the encysted form of the parasite is eaten, the capsules of the cysts are dissolved and the trichinae set free. The larvae burrow into the mucosa of the small intestine. By the seventh or ninth day, hundreds of embryos are produced. They then travel via the intestinal lymphatics to the circulatory system and thence to the muscles where they penetrate the muscle fibres and cause myositis. A capsule develops around the embryo in about six weeks, and later on, lime salts are deposited in the capsule. The incubation period may vary from two to thirty days, but more often than not, it is about one week.

In our case, there appeared two possible routes as to how the disease was contracted. Since the patient admitted having frequented Chinese restaurants for pork dishes, one could very readily consider this avenue the most logical. However, she also admitted, later that very often she would prepare near-raw hamburgers in her own kitchen. The N. Y. City Health Department was inclined to believe that the latter route was the more probable because often a butcher will grind pork for one customer and then in the same meat grinder, without cleaning it, he will grind beef for another. It was pointed out that similar cases had been traced to this origin in the past in the east mid-Manhattan district, where our patient lived.

Since some of the abnormal neurological findings in CNS trichinosis are mild and fleeting, and since some are severe and lasting as in Sprink's case which resulted in a permanent right hemiplegia,[10] it would seem as though these signs could be caused by a toxemia, by a migration into the brain by the larval parasites, or by both.[11] That such a migration is possible can be proven by simply comparing the diameter in cross section of a larva which happens to be 6u with that of an erythrocyte which happens to be 7u. According to Tice, one should expect to find an intense lymphocytic and leucocytic meningeal reaction and a similar exudate in the perivascular spaces of the cerebral blood vessels. One should also expect to find trichinae embryos in the brain walled off by glial cells, and degenerative changes in the ganglion cells.[12] However, in the second case of Houston and Ross which ended fatally, sections of the frontal, parietal, temporal, and occipital cortex, antrum, semi-ovale, basal ganglia, pons, medulla, and cerebellum, showed neither larvae or inflammatory foci.[8]

As a matter of historical interest, we may add that T. Spiralis was first demonstrated in the brain in 1906 by Frothington,[13] and was first demonstrated in the cerebrospinal fluid in 1914 by Van Cott and Lintz.[14]

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Neurological Clinical Pathological Conference

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CHARLES D. ARING, M.D., *Editor*

CASE NO. 237059

Presentation

On June 27, 1951, a 39-year-old colored man was brought to the hospital because of headaches of increasing severity. The patient was lethargic, somewhat confused, and unable to give his story. His wife stated that he had been fairly well until about two weeks prior to admission when he began to complain of headache. He developed fever, sweats, and general malaise. His fellow workers noted that he had been sluggish and had difficulty in performing his usual occupation (laborer in manufacture of roofing material). His past history was relevant in that he was known to have had bronchial asthma and severe recurrent sinusitis of the maxillary frontal and ethmoid sinuses.

Initial physical examination revealed that he was moderately ill, quite lethargic, and able to cooperate only slightly. Blood pressure was 130/85, pulse 88, respirations 20, and rectal temperature 101°F. On auscultation of the chest there was moderate wheezing but no dullness. The heart sounds were of good quality. There was mild weakness in both right extremities and a central type of right facial weakness. Within the limits of his ability to cooperate, tests of coordination were well-performed. The deep reflexes were equal on the two sides. The appreciation of pain and vibration were normal. Examination of the cranial nerves revealed external strabismus. The pupils were normal. No papilledema was noted. There was no evidence of upper respiratory infection at the time of admission.

Lumbar puncture was performed shortly after admission and subsequently (see table). Total white blood cell count was 7,600 per cmm., differential count revealed 42 per cent neutrophils, 48 per cent lymphocytes, and 10 per cent monocytes. Red blood count showed 4.7 million cells. Routine urinalysis was normal. Blood Kahn was negative. Roentgen examination at the time of admission showed clouding of the frontal sinuses. Chest film was normal except for minimal infiltrate of the extreme left apex which was believed to contain some calcium. Culture of gastric washings failed to grow an organism.

On the day after admission (June 28) the patient was less responsive and cooperative than he had been previously. Although he was being treated with 300,000 units of penicillin and 0.5 grams of aureomycin every 4 hours, his rectal temperature remained mildly elevated to 100° F. It was believed that he had an extensor plantar response on the right; the remainder of the examination remained as it had been on admission.

On July 1 the right pupil was 3 mm. in diameter and reacted well to light; the left pupil 2 mm. and reacted poorly. There was no elevation of the optic discs. Electroencephalogram revealed a prominent left frontal slow-wave focal pattern. Accordingly, ventriculogram was decided upon, which showed minimal deformity, suggestive of a depression of the right lateral ventricle. Craniotomy in the right fronto-parietal area revealed nothing abnormal. The patient withstood the procedure poorly. For several days following operation the patient remained in poor condition, responding only to painful stimuli. He then seemed to improve somewhat and was placed on 250 mg. of streptomycin every four hours in addition to penicillin and aureomycin. On July 10, 50 mg. of streptomycin (to a total of one gram) and 10,000 units of penicillin were injected intrathecally. Streptomycin (500 mg. intramuscularly every 12 hours) was continued until a total of 30 grams had been administered. He had received 20 grams of aureomycin.

The patient did poorly, never really regaining contact with the environment. He continued to have a right hemiparesis. On July 29 the bladder was found to be severely distended. It was impossible to establish adequate drainage by catheter because of intravesical bleeding and clot formation. A suprapubic cystotomy was done under local anesthesia. He was placed on tube feedings. The patient continued to run a mild, low-grade fever (rectal temperatures of approximately 101°F.). Small collections of fluid under the operative flap were removed from time to time. On August 19 he vomited after tube feeding, had obvious difficulty breath-

CEREBROSPINAL FLUID

Date	Initial Pressure	CC Removed	Final Pressure	Polys/ Cmm.	Lymphs/ Cmm.	Protein Mg. %	Sugar Mg. %	Chloride Meq./liter
6/27	240	10	120		80	96	50	115
6/28	135	10	110		98	96	50	
6/30	160	5	130	48	79	129	50	
7/1	Ventricular fluid					14	87	111
7/4	450	20	200			52		
7/10	200	10	150			84	21	107
7/14	50	10	0			117	19	101
7/23	60					192	29	110
7/27	110	10	110	49	16	140	18	101
7/31	10				60	150	54	112
8/3	50					236	27	97
8/19	600+ Grossly bloody							

ing, and developed coarse rhonchi over both lungs. He was placed in postural drainage position and required frequent suction. The temperature rose to 104° rectally. Lumbar puncture revealed grossly bloody fluid under 600+ mm. pressure. A catheter was inserted into the subarachnoid space. He seemed to improve slightly, but on the afternoon of August 20, 1951, he died suddenly.

Nine additional lumbar punctures were performed for the intrathecal administration of streptomycin. Five attempts to demonstrate organisms by smear and culture were unsuccessful. A guinea pig injected with csf on July 10 showed no tuberculosis at autopsy on September 12. Wassermann and colloidal gold reactions were negative on several occasions.

Differential Diagnosis

*Dr. Morton Hamburger**: The case that we have under discussion presents some interesting clinical problems, problems of decision at the bedside. The protocol is well written and makes it easy for anyone reading it to project himself back to the bedside and to share with the physicians who were responsible for this patient's care some of their anxieties and wonderings. He was a 39-year-old colored laborer who was brought to the hospital by his wife. She had to give the history because the patient was unable to do so. He had not been ill long, but for some two weeks prior to admission suffered from frontal headaches which apparently had become more severe as time went on. In addition, he had fever and sweats and general malaise, and his fellow workers observed that he was sluggish although they evidently did not notice anything specific about this sluggishness.

His wife stated that he had had bronchial asthma for many years but I don't believe that plays any role in the illness that brought him to the autopsy table. In addition, he had recurrent, severe sinusitis that might have had something to do with the illness that killed him. We certainly need to discuss it.

The initial physical examination showed him to be stuporous—lethargic is the word used—and he was able to cooperate but slightly. His blood pressure was normal; he had a fever of 101° F. His pulse was a little fast, and his respirations were not described as remarkable. Not a word is said about his neck, which may or may not have been stiff; certainly this was noted at the bedside. At any rate, the patient did have definite signs of right hemiparesis, as evidenced by weakness of both the arm and the leg and facial weakness; later it was thought likely that he had a Babinski on that side. Other than the hemiparesis, the neurological examination revealed little that is diagnostically helpful. He had no papilledema. We are led to suspect that this young man had a space-occupying lesion on the left side of the brain, and further evidence as we go along tends to support that notion but not prove it.

Various laboratory studies were done in an attempt to elucidate this patient's illness. To begin with, he had a normal white count although the differential count revealed some relative increase in his lymphocytes up to 48 per cent. He was not anemic. His Kahn was negative, and that is of some importance. He had x-rays of his chest and of his sinuses. The x-rays were described as showing clouding of the frontal sinuses and minimal infiltrate at the apex of the left upper lobe of the lung. Therefore, tuberculosis was suspected. A tube was dropped into his stomach, the washings were cultured, but no bacteria grew out. From that I presume is meant that there were no turbercle bacilli. Other laboratory studies were done, and the most important of course were those related to his central nervous system. He had an electroencephalogram which showed a slow rhythm on the left side conpatible again with a space-occupying lesion. In addition to that, he had ventriculograms. Perhaps a member of the Radiology Department might comment on them at this point.

Dr. Emmanuel Levin: Our report noted enlargement of the lateral ventricles with incomplete filling of the right lateral ventricle. There was no shift of the ventricular system. The flattening of the long segment of the right lateral ventricle seen in the lateral view was believed due to insufficient filling. If there was a large tumor in this area, there would not only be the incomplete filling but shift of the lateral ventricle to the opposite side. In a film in the brow-up position there was no evidence of depression and there was equal filling in portions 1 and 2 of both ven-

*Associate Professor of Medicine.

tricles. The roentgenologic diagnosis was slight to moderate enlargement of the lateral ventricles with no definite evidence of a space-consuming lesion.

Dr. Hamburger: Evidently there was some enlargement of the lateral ventricles which perhaps may have been due to a degree of hydrocephalus, possibly due to a block or a partial block at the base of the brain.

The spinal fluid was examined on numerous occasions, and I should like to analyze what was found. The pressure originally was elevated; it was 240 mm. of water, and after that it fluctuated within fairly wide limits. It was as low as 10 mm. on one occasion, and excluding the final tap, was as high as 450 mm. The latter, of course, is a distinct elevation provided that the patient is relaxed, and there is nothing to indicate that he was anything but relaxed during his entire course. He had a constant pleocytosis. Usually the cells showed a preponderance of lymphocytes, but this preponderance was not always great. On one occasion toward the latter part of his course there was actually a preponderance of polymorphonuclear leukocytes. The cell counts were never high, never in the range of purulent meningitis. They were always in the range of chronic meningitis which we are going to have to consider. Certainly the protein was elevated consistently. It varied over not too wide limits, ranging from a low of 52, and I exclude the one ventricular fluid that is reported, up to 192 mg. per cent.

Now from the standpoint of differential diagnosis, perhaps the most important chemical findings in the cerebrospinal fluid are the sugar and chloride values. The sugar was reported at 50 mg. per cent on admission, which is normal, and yet as we peruse the sugar values we find that with the exception of one of 54 mg. per cent on August 3, the remainder were all low. That is, they ranged from 19 to 29 mg. per cent, and we must assume that these sugars were determined promptly after the cerebrospinal fluid was withdrawn. If this is the case, these are significantly abnormal values. The chloride values are also low, though I don't think they are quite as helpful from a diagnostic standpoint as the sugar determinations. Normal chlorides in the cerebrospinal fluid expressed in milliequivalents is 118 to 126 per liter; this patient never had at any time chlorides that were as high as the lower limits of normal, and at one time they were recorded as low as 101. So I would be inclined to accept these low chlorides also as being of some significance. In addition to these positive findings, we have some negative findings which are helpful. The Wassermann was negative. The spinal fluid was cultured and was smeared on numerous occasions and neither the smears nor the cultures ever yielded bacteria.

The patient's course was inexorably downhill. He improved only slightly at one time. He ran a low grade fever, and, though he was treated with three different antibiotics, namely, penicillin, streptomycin, and auremycin, he continued to lose ground, and he finally died nearly two months after he was admitted to the hospital. Well, what did he have? I believe that he had a small space-occupying lesion, though he needn't have because these findings could have been due to vascular troubles. The electroencephalogram is suggestive of a small space-occupying lesion even though the air studies are not. He could have had a tuberculoma. He may have had tuberculous meningitis. Tuberculous meningitis, particularly when the course is prolonged by treatment, may produce changes in the arteries. In untreated tuberculous meningitis, in my experience, it is uncommon to have hemiparesis. If the patient exhibits weakness of extremities, the weakness usually is that associated with debilitating illness and not with a specific neurological lesion. The fact that he did have hemiparesis makes me think that if this patient had tuberculous meningitis, he had, in addition, a tuberculoma in an area where it could produce this effect.

Also we have to consider gumma. I don't think we need to consider it very seriously, but in an exercise of this sort and also at the bedside we must always consider certain things which we do not linger over very long, and gumma of the brain is one of these. I don't believe there is much evidence for syphilis. It is well to mention the possibility that this could be late meningovascular syphilis. From a strictly clinical standpoint, that is an acceptable diagnosis, but not one

which strongly urges itself upon us. Even if this man had a positive Wassermann reaction I don't believe I would have been willing to make a diagnosis of syphilis of the central nervous system as responsible for this final illness.

He could conceivably have had a brain tumor. A glioma of some sort might have accounted for these findings. I find it difficult to explain the cerebrospinal fluid findings on this basis and particularly the gradually lowering sugar and the low chlorides. I don't believe we should have these laboratory findings had this patient a glioma, and I don't believe that a benign tumor can in any way account for this illness.

Could he have had a brain abscess? He had pansinusitis over the years, and we know that the sinuses are a fruitful source of brain abscess. Brain abscesses can arise by direct extension particularly from the frontal sinuses and they can run a remarkably quiet course. Sometimes a brain abscess can exist without any fever or leukocytosis. We've seen such cases here and they have been reported by others. Here again I come back to the cerebrospinal fluid and find it difficult to account for the findings on the basis of a brain abscess. Though I believe we could certainly account for the cellular changes, for the pressure, and probably for the protein on the basis of a brain abscess lying fairly close to the surface, the sugar and chloride values would be more difficult to explain. This would be the so-called sympathetic reaction to such an abscess, but unless there are bacteria present in the cerebrospinal fluid itself, I find it difficult to explain the sugar and the chlorides. So I don't believe that a brain abscess satisfactorily accounts for this patient's illness.

The two rare things that crossed my mind as I read this protocol were the possibility of cerebral schistosomiasis which occurs in returning veterans from time to time, and the possibility of a fungus infection of the central nervous system, torulosis. I see no reason to consider schistosomiasis at all seriously. Torulosis perhaps might be considered a little more seriously, but these patients usually don't die so quickly. They go on, the illness lingers, and they may live for several years with a low grade chronic infection that is finally fatal. The relatively slow drop in the cerebrospinal fluid sugar is compatible with this diagnosis, but here again we must accept at its face value the statement that no organisms were seen in the fluid. Torulae are very easy to see in cerebrospinal fluid, and any alert observer will pick up the budding. Usually what happens is that a diagnosis of lymphocytic meningitis of some kind or another is made, and someone notices that the lymphocytes are sprouting and then the diagnosis becomes clear, but I don't believe we need dwell on that.

These, then, are the important diagnostic considerations. There are one or two other matters which may have some bearing on the diagnosis though I'm not sure how important they are. The appearance of bloody urine is not easy to explain unless the patient had had an indwelling catheter for some time. His admission urine was reported as normal. He could have had tuberculosis of the bladder, that is not an unusually rare lesion. The episode of bloody urine could be accounted for on that basis. But if he had tuberculosis of the bladder, I think it somewhat unlikely that he would not have had pus in his urine when he first was seen at the hospital several weeks before bloody urination. So perhaps it isn't too fantastic to imagine that he had had an indwelling catheter for some time which had irritated the bladder and perhaps accounted for the blood.

The mode of his death is interesting. Obviously something gave way suddenly. He died with bloody cerebrospinal fluid, with increased intracranial pressure above 600 mm. A blood vessel gave way, whether on convexity in the mass of exudate or at the base of the brain, I don't know. Any space-occupying lesion ultimately may erode a blood vessel and cause death. I would guess that the lesion that led to the blood in the cerebrospinal fluid was probably at the base of the brain rather than in or about the ventricular system. Of the possibilities, I think those to be seriously considered are tuberculosis of the central nervous system and possibly brain abscess. From a strictly diagnostic standpoint, it seems to me that the crux of diagnosis in this case is particularly

the level of the sugar and of the chlorides in the cerebrospinal fluid. If there had been only one low sugar determination along with the rest of this clinical picture the debate on diagnosis would have been far more prolonged. This series of low sugar values spell tuberculous meningitis to me despite the fact that the tubercle bacillus was not demonstrated. I think he may also have had a small tuberculoma in the left side of his brain which was responsible for his hemiparesis; it is unlikely that he may have had tuberculosis of the bladder. The terminal cerebral hemorrhage I consider to be a complication of the tuberculous process.

Dr. Murton Shaver: Was there any sign of weakness in the left leg or on the left side of the body?

Dr. Samuel A. Trufant: No, terminally he was generally weak; the hemiparesis remained on the right.

Dr. Daniel Osher: Do you know if manometric cerebrospinal fluid studies were done? A block is suggested by the fact that the cerebrospinal fluid pressures at times were as high as 450 mm. and at other times were as low as 50 and 10.

Dr. Aring: Manometrics were done on the taps June 27, 29, 30, and July 4. These were reported to be normal.

Dr. Luther Martin: Regarding the low pressures, he was draining cerebrospinal fluid from under the flap, therefore, the low pressure reading may not be representative.

Dr. Ralph Kahana: Were there any convulsions?

Dr. Trufant: None that I recall; there was no record of any.

Dr. Kahana: My impression is that convulsions are seen in all or almost all cases of tuberculous meningitis.

Dr. Hamburger: That has not been our experience; my impression is that we see convulsions in a minority of cases here.

Dr. Trufant: I agree that is the case in adults.

Dr. Hamburger: Our experience, of course, has been with adults. In children fits may be common in tuberculous meningitis, but I doubt it.

Discussion of Pathology

Dr. Murton Shaver: The autopsy was performed 1½ hours postmortem. In addition to the central nervous system findings the remainder of the pathological lesions consisted of acute bronchitis with pneumonia of the lower lobes bilaterally, tuberculous adenitis at the apical lymph nodes with pleural adhesions, and a partial atelectasis of the right middle lobe. In addition, a suprapubic cystotomy was present, and there was an acute cystitis. He also had a decubitus ulcer over the buttocks.

The brain weighed 1550 grams and showed generalized flattening of the convolutions. The leptomeninges were generally thick over the convolutions and a fungus was present at the site of the operation. Here the cortex protruded approximately 1½ cm. beyond the bony defect in the skull. There was no evidence of herniation across the midline under the flax. In the median cleft, above the anterior part of the corpus callosum a few

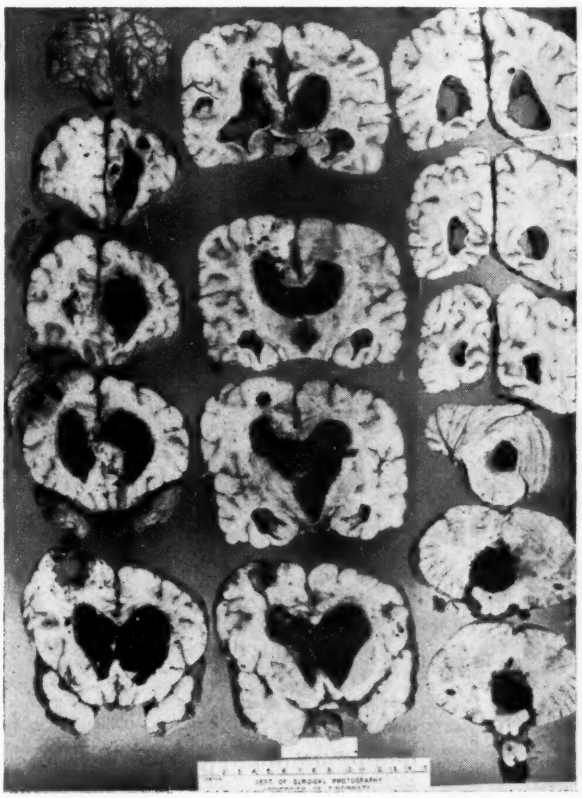

Figure 1. A photograph of the vertically sectioned brain attempting to show: (1) the casting of the ventricles with clotted blood, (2) the origin of the bleeding in the left frontal lobe, (3) the softening and disintegration of the mesial face of *both* frontal lobes, and (4) the operative site, overlying the right fronto-parietal area.

small white nodules (probable tubercles), were observed grossly, measuring up to 2 x 3 mm. in diameter. These were confined to the mesial aspects of both frontal lobes. Elsewhere nothing of this sort was observed. Also on palpating in these mesial areas the superficial cortex felt soft anteriorly above the corpus callosum. The inferior surface of the brain was not remarkable aside from a small erosion of the superficial cortex in the inferior left frontal region, along the gyrus rectus, and for the presence of a thin film of blood clot over the lower brain stem in the region of the lower pons and medulla.

Coronal sections made through the cerebrum and brain stem and cerebellum failed to show any displacement of the midline structures (Figure 1). The ventricles were markedly dilated and filled with blood clot in the form of a cast. In the left frontal region mesially there was an area of erosion of the ependymal surface, and here a cavity was found on dissecting this tissue infiltrated with hemorrhage. This cavity measured approximately 3 cm. in diameter and extended toward the medial aspect of the left frontal lobe. The blood clot extended throughout the lateral, third, and fourth ventricles. It appeared that the blood lying under the base of the brain had come by extension from the ventricles; also it appeared from the distribution of clot that the point of bleeding lay in the left frontal region. This region was carefully dissected, exposing the left anterior cerebral artery with an aneurysm lying opposite the anterior portion of the corpus callosum. The aneurysm presented a point of rupture, and the bleeding had spread in the frontal lobe and had dissected into the ventricle. Both anterior cerebral arteries were firm and surrounded by fibrinous exudate, and the walls appeared thickened grossly.

There was no evidence of basal meningitis grossly, or microscopically either except for the presence of a few scattered cells. Over the surface of the brain elsewhere there were scattered lymphocytes and endothelial cells but no definite masses such as I have demonstrated. Inflammatory changes were minimal. The leptomeninges were markedly thickened and fibrosed over some areas as though there may have been healing in an old leptomeningitis. The final diagnosis as regards the central nervous system is as follows: A congenital aneurysm of the left anterior cerebral artery which ruptured; encephalomalacia in the left frontal lobe due to this hemorrhage with extension into the entire ventricular system. There was a treated, minimal, tuberculous meningitis, most marked over the medial aspects of both frontal lobes and septic thrombosis of the anterior cerebral arteries bilaterally; encephalomalacia of the mesial aspects of both frontal lobes due to thrombosis of both anterior cerebral arteries; and post-operative right parietal osteoplastic craniotomy with a cerebral fungus.

Clinical Diagnosis

Tuberculous meningitis.
Softening of the cingulate gyrus, right.
Aneurysm of circle of Willis with rupture.

Dr. Hamburger's Diagnosis

Tuberculous meningitis.
Tuberculoma, left cerebral hemisphere.
Rupture of vessel at the base (circle of Willis).

Anatomical Diagnosis

Congenital aneurysm of the left anterior cerebral artery, ruptured with hemorrhage filling the ventricles.

Encephalomalacia due to hemorrhage, left frontal lobe.

Tuberculous meningitis, treated, minimal, most marked over the medial aspects of both frontal lobes.

Septic thrombosis of the anterior cerebral arteries bilaterally.

Encephalomalacia, mesial aspects of both frontal lobes due to thrombosis of both anterior cerebral arteries.

Post-operative right fronto-parietal osteoplastic craniotomy with a cerebral fungus.

Bronchopneumonia.

Partial atelectasis of right middle lobe of lung.

Dr. Osher: This case brings up a problem that is not limited to tuberculous meningitis but has been called to my attention particularly in acute purulent meningitis. Seemingly new syndromes have been developing since the advent of potent drugs in the treatment of meningitis. While I'm not too familiar with the literature about it I know we have seen cases in which there seemed to be cure of the acute phase of meningitis and yet neurological dis-

ease would progress. At autopsy there would be varying types of lesions, sometimes obstructive hydrocephalus due to scarring, sometimes multiple areas of softening associated with vascular changes similar to those in today's case. Apparently in the past some of these cases would have died quickly without the antibiotics and are now being kept alive to develop these secondary changes in vessels and in the other tissues probably as a result of organization. In this case of tuberculous meningitis the patient apparently responded to treatment, but developed these vascular complications.

I would like to ask Doctor Shaver if it is not possible that this hemorrhage might represent that from some form of mycotic aneurysm of the anterior cerebral artery secondary to tuberculosis of its wall?

Dr. Shaver: I expected that question and have made inquiries among pathologists and neuropathologists. No one has seen such a lesion in tuberculosis. The nature of this lesion is in keeping with a congenital aneurysm. It is possible that its rupture in some manner may have been precipitated by the inflammatory infiltration.

Dr. Trufant: Dr. Shaver, was there at brain cutting any evidence of an older gliotic scar in the left frontal lobe?

Dr. Aring: You mean older than in the right? Scars were found in both.

Dr. Trufant: No, I mean a scar deep in the substance as though the aneurysm had ruptured previously.

Dr. Shaver: I couldn't tell.

Dr. Trufant: I wondered if a leak prior to the terminal event might have been responsible for the hemiparesis found on admission?

Dr. Shaver: Such evidence would have been obliterated by the second one.

Dr. Osher: Was there any ependymitis? Was the aqueduct dilated and was there hydrocephalus?

Dr. Shaver: The ventricles were dilated throughout with blood clots. There was no ependymitis.

Dr. Hamburger: Dr. Shaver, isn't this an unusual location for congenital aneurysm?

Dr. Shaver: Yes, but they occasionally do occur intracerebrally.

Dr. Hamburger: Was there any pathological explanation of the electroencephalography findings?

Dr. Shaver: These findings in connection with the aneurysm, were some evidence that it had previously wept or bled, but I can only assume that.

Dr. Hamburger: Do you think that without the aneurysm he would have recovered?

Dr. Shaver: That I can't answer.

Dr. J. P. Evans: It is important to get to the matter of therapy. Dr. Shaver, would you comment on the age of the lesion in the frontal lobe not associated with the aneurysm.

Dr. Shaver: I would postulate between one month to three months

Dr. Evans: I would like to point to the preoperative note which was written on July 1 and he died seven weeks later on August 23. "He is a colored man, a 39-year-old asthmatic of long duration who is said to have had a pulmonary acid-fast lesion. There is no known lues. Examination today shows a drowsy, only slightly responsive individual whose intellectual and sensory capacities cannot be determined. The fundi are normal, the physiologic cups being deep and disc color normal. The right pupil is 3 mm. and reacts well to light; the left pupil, 2 mm., reacts poorly. There is left external squint. There is a drifting downward of the right arm. Abnormal skin reflexes are absent. The plantar responses are flexor bilaterally. The electroencephalogram has shown a focus in the left frontal region. In view of the increasing drowsiness and the rather minimal focal signs, I think he would require ventriculography to rule out a left frontal expanding lesion. Lues may be the etiologic factor, but I now think this unlikely and do not believe we should wait any longer for the Wassermann report. Since he has not improved in 72 hours with penicillin and aureomycin, abscess would seem unlikely. In view of the lack of increased intracranial pressure to date, I would favor tuberculoma. On this basis it might be well to change to streptomycin in the case tuberculoma were uncovered at operation."

Well, he deteriorated during the day (July 1) so ventriculograms were done that same night and these we interpreted as showing a depression of the right lateral ventricle. He was taken to the operating room with a diagnosis of possible parasagittal meningioma on the right.

The next note is a pre-operative one which records that ventriculograms, while not too definite, suggested the possibility of a frontal lesion along the falx. Coupled with this was the clinical state which suggested to Dr. Luther Martin cingulate gyrus involvement as has been described by Dr. J. M. Nielson. We recognized that this might be an infectious process, but did not feel justified in withholding operation. I merely wanted to note that there had been suspected a lesion in the cingulate region where Dr. Shaver had demonstrated it.

Dr. Aring: Those are clarifying points and Dr. Martin shuld be complimented on exquisite localization. Dr. Hamburger, would you comment on the therapy? Was it adequate?

Dr. Hamburger: First, I'd like to ask if the neck was stiff.

Dr. Trufant: No, not that I recall at any time. Maybe Dr. Martin who saw him later might answer.

Dr. Martin: Eventually it became very stiff.

Dr. Aring: In the early examinations it was recorded as supple.

Dr. Hamburger: When this patient was first seen there must have been a serious therapeutic dilemma because there was a diagnostic problem. Here was a man who had a hemiparesis, without having a stiff neck and with no diminution of sugar in the cerebrospinal fluid. I don't believe that the diagnosis of tuberculous meningitis could have been made on admission and I would be the last to criticize anyone who did not give the patient strepto-

mycin. In general, streptomycin is not an antibiotic that one uses without due consideration because when it is used over a period of time, it can and often does produce toxic effects, notably on the vestibular portion of the eighth nerve. as we all know. It was not until the patient had been in the hospital two weeks that tuberculous meningitis was seriously considered. Whether he could have gotten well in the absence of a subarachnoid hemorrhage is a matter of speculation.

The treatment of tuberculous meningitis is not very satisfactory. There are patients who are doing well, and during the 4 or 5 years that streptomycin has been available in clinical practice, we have learned much as to its use, but the last word is far from having been said. Right now most of us feel that the patient with tuberculous meningitis must be treated for a year with antibiotics. Streptomycin should be given intramuscuarlly during that time and in the last couple of years we have learned that the addition of paraminosalicylic acid has made a difference. One could criticize the management of this case because paraminosalicylic acid was not given. I would be willing to say that it wouldn't have made any difference in the course of this man's disease as it developed, because the chief role of this drug is to interfere with the development of streptomycin-resistant tubercle bacilli. During the period of time under discussion, it is not likely that that streptomycin-resistant bacteria will have appeared.

The question of the intrathecal use of streptomycin is a debatable one. I share Doctor Aring's oft-stated opinion that the less we put into the subarachnoid space, the better off the patient will be. Right now, however, I do feel that what evidence we have impels us to continue to use intrathecal streptomycin daily in tuberculous meningitis for about 2 months. Our only two good results on the medical service have been achieved with this regimen, perhaps in spite of it. At the Fitzsimons General Hospital in Denver, an Army Hospital where tuberculosis patients are sent, they have been exploring the possibility of treating tuberculous meningitis without intrathecal streptomycin and are encouraged with their results. We may come around to that here. There are other new methods which require evaluation and more study.

There is one other problem for which we have no answer at the moment, and that is the vascular lesion of tuberculosis. Whether antibiotics have a beneficial effect on the arteritis, I do not know. A number of pathologists have reported that as the tuberculous lesion heals, in some cases the shrinking of scar tissue tends to constrict vessels and to produce infarction of the brain with hemiplegia and other signs secondary to vascular change. At the present time this is an unsolved problem. To what extent the healing process itself does this, I don't know. In this case there is plain evidence, as we have known over the years, that there was an arteritis present, most assuredly before treatment was commenced.

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